

Decision Memo for Continuous Positive Airway Pressure (CPAP) Therapy for Obstructive Sleep Apnea (OSA) (CAG-00093N)

Decision Summary

CMS will revise the NCD for CPAP for the treatment of OSA (CIM 60-17) to the following: CPAP will be covered under Medicare in adult patients with OSA if either of the following criteria is met:

(1) AHI > 15, or

(2) AHI > 5 and > 14 with documented symptoms of excessive daytime sleepiness, impaired cognition, mood disorders or insomnia, or documented hypertension, ischemic heart disease or history of stroke.

The AHI is equal to the average number of episodes of apnea and hypopnea per hour and must be based on a minimum of 2 hours of sleep recorded by polysomnography using actual recorded hours of sleep (i.e. the AHI may not be extrapolated or projected). Two hours of recorded sleep is consistent with current practice. Apnea is defined as a cessation of airflow for at least 10 seconds. Hypopnea is defined as an abnormal respiratory event lasting at least 10 seconds with at least a 30% reduction in thoracoabdominal movement or airflow as compared to baseline, and with at least a 4% oxygen desaturation. The polysomnography must be performed in a facility-based sleep study laboratory, and not in the home or in a mobile facility.

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Decision Memo

To: Administrative File: CAG 00093C
Continuous Positive Airway Pressure (CPAP) Therapy Used in the Treatment of Obstructive Sleep Apnea

From:

Sean Tunis, MD, MSc
Director, Coverage and Analysis Group

Jeffrey Shuren, MD, JD
Director, Division of Items and Devices

Francina C. Spencer
Health Insurance Specialist, Division of Items and Devices

Joseph Chin, MD, MS
Medical Officer, Division of Medical and Surgical Services

Jacqueline Sheridan
Technical Advisor, Division of Items and Devices

Subject: Coverage Decision Memorandum for CPAP

Date: October 30, 2001

This memorandum serves several purposes: (1) provides a brief clinical background on obstructive sleep apnea, (2) reviews the history of the Medicare policy for CPAP, (3) reviews and analyzes relevant scientific and clinical literature on the use of positive airway pressure devices, (4) delineates the reasoning for announcing our intention to review the national coverage policy on CPAP, and (5) delineates the reason for and announces our decision to revise the current national coverage determination (NCD).

Clinical Background

Sleep apnea refers to a collection of conditions and syndromes that have periods of apnea, a temporary cessation of breathing, as key occurrences. It was initially described in the early 1800's. One of the first accounts was written by Charles Dickens in 1837 and entitled *The Posthumous Papers of the Pickwick Club*. Subsequently, William Osler coined the term "Pickwickian" to describe the obese, hypersomnolent patient in 1918. Over the years, various sleep apnea syndromes have been described and classified into three main types: central, obstructive, and mixed. Central sleep apnea refers to apnea syndromes with origins in the central nervous system. Obstructive sleep apnea (OSA) refers to apnea syndromes due primarily to collapse of the upper airway during sleep. Mixed apnea refers to apnea with both central and obstructive characteristics. ¹

Of the three main types of apneas, OSA has received the most scientific interest and study. The prevalence of OSA in the United States has been estimated to be about 2-4% of middle age adults.² OSA has also been identified as a risk factor for hypertension.^{3,4,5} The pathogenesis and pathophysiology of OSA has been studied extensively. During sleep, the upper airway becomes occluded, resulting in an episode of apnea. As a result of the apnea, the patient experiences a brief arousal from sleep. With the return of breathing, the patient typically returns to sleep quickly. This sequence is repeated over and over. The pharynx has been identified as the primary site of obstruction in most patients. A number of anatomical and functional factors, such as negative oropharyngeal pressure, decreased muscle activity, and possible narrowing of the oropharyngeal lumen, may also be involved in the collapse of the upper airway during sleep. ⁶

Symptoms of OSA include somnolence, fatigue, irritability, headaches, cognitive impairment, depression, and personality changes.⁷ There are a number of medical and surgical treatment options for OSA.^{8,9} Nonpharmacologic medical treatments include weight reduction, tongue-retaining devices, positive airway pressure modalities such as continuous positive airway pressure (CPAP) and bilevel positive airway pressure (BiPAP). CPAP involves the administration of air usually through the nose by an external device at a fixed pressure to maintain the patency of the upper airway. BiPAP is similar to CPAP but the devices are capable of generating two adjustable pressure levels. Medications that may be used in OSA include oxygen, protriptyline and theophylline. Surgical procedures include uvulopalatopharyngoplasty, somnoplasty and tracheostomy.

While the occurrence of apnea has remained a primary diagnostic criterion for sleep apnea, episodes of reduced ventilation have received considerable attention and clinical consideration since the 1980's. The term hypopnea has been used to describe these episodes of reduced breathing; however, there was no general consensus for the definition of hypopnea at the time.¹⁰ Variations in the definition of hypopnea still persist today. Despite such variations, the apnea-hypopnea index (AHI = number of episodes of apneas and hypopneas per hour of sleep) has been utilized extensively in recent years in the published literature in the definition of OSA. The AHI has also been called the respiratory distress index (RDI). Hypopneas and the AHI will be discussed in greater detail later in this decision.

History of Medicare Coverage

In 1986, the Centers for Medicare and Medicaid Services (CMS - then known as the Health Care Financing Administration) requested the Office of Health Technology Assessment (OHTA) to conduct an assessment of the safety, clinical effectiveness and use of CPAP. OHTA reported that "the consensus of clinical opinion from the available information appears to be that CPAP can in the majority of cases prevent OSA and provide substantial clinical improvement with minimal associated morbidity." They went on further to recommend that "the use of CPAP be covered under Medicare when used in adult patients with moderate and severe OSA who have failed to obtain relief from other non-invasive therapies and for whom surgery would be the only other therapeutic alternative."¹¹ The diagnosis of OSA required at least 30 episodes of apnea, each lasting a minimum of 10 seconds, during 6-7 hours of sleep. These specifications were based predominately on expert opinions at the time.¹²

Based on the OHTA technology assessment, Medicare issued a national coverage determination (see CIM 60-17) which covered CPAP for adult patients with moderate or severe OSA for whom surgery is a likely alternative (effective date January 12, 1987), and adopted OHTA's recommendations on the diagnosis of OSA. Since the 1986 decision specifically addressed CPAP only, the Durable Medical Equipment Regional Carriers (DMERCs) have issued a respiratory assist devices regional medical review policy (RAD RMRP) that addresses BiPAP devices and other accessories (last revised in 1999). Specifically for the treatment of OSA, a respiratory assist device with bilevel pressure capability, without backup rate feature, used with noninvasive interface (K0532) will be covered for the first three months of noninvasive positive pressure respiratory assistance (NPPRA) if the following criteria are met:

1. complete facility-based, attended polysomnogram has established the diagnosis of obstructive sleep apnea, and
2. single level device (E0601, CPAP) has been tried and proven ineffective.¹³

Timeline of Recent Activities

- May 21, 2000 CMS met with the American Academy of Sleep Medicine (AASM). AASM asked us to revise the national coverage policy and to include allowance for hypopneas in the diagnosis of patients with moderate or severe OSA.
- June 4, 2001 CMS received a request for a revised national coverage determination.
- July 24, 2001 CMS met with representatives of the American Academy of Sleep Medicine (AASM). AASM believes that the current national coverage determination needs to be revised.
- August 23, 2001 CMS participated on a DMERC teleconference to discuss the CPAP issues.
- September 4, 2001 CMS received additional materials for review from device manufacturer and professional organizations.

FDA Approval/Clearance

CPAP, BiPAP and related devices have been considered and cleared for marketing by the Food and Drug Administration (FDA) under a 510(k) process. The 510(k) is a notification of intent to market a specific device. The FDA has determined that certain CPAP and BiPAP devices are "substantially equivalent to legally marketed predicate devices marketed in interstate commerce prior to May 28, 1976, enactment date of the Medical Device Amendments, or to devices that have been reclassified in accordance with the provisions of the Federal Food, Drug, and Cosmetic Act." A substantially equivalent determination assumes compliance with the Good Manufacturing Practice requirements, as set forth in the Quality System Regulation (QS) for Medical Devices: General regulation (21 CFR Part 820) and that, through periodic QS inspections, the FDA will verify such assumptions. Failure to comply with the GMP regulation may result in regulatory action. Typically, no clinical data is required as part of the 510 (k) application, but instead the clearance process focuses on technical performance. There are currently 167 FDA approved 510(k) CPAP and related devices for use in the home on the market.

Summary of Evidence

Since 1986, there have been numerous studies published on OSA and CPAP. The scientific evidence, knowledge base and standards of practice have evolved considerably. Accordingly, CMS was asked to reevaluate the coverage policy for CPAP and the diagnostic criteria for OSA by the DMERCs. In this reevaluation, CMS focused on the following questions:

1. What diagnostic criteria for moderate to severe OSA are currently used?
2. Should hypopnea be included in the diagnostic criteria of OSA?

3. Is there scientific evidence of the effectiveness of bilevel positive airway pressure (BiPAP) for the treatment of OSA?

What diagnostic criteria for moderate to severe OSA are currently used to determine treatment with CPAP?

As noted earlier, the use of the AHI in determining when to treat OSA has become common in the reported literature. However, the threshold level used for initiating treatment has varied across studies. To better understand current practices, Medline (mid 1998 to 2001) was searched using the keywords obstructive sleep apnea (OSA) and CPAP separately and in combinations. Over 100 citations were found. The literature search was then focused based on the following criteria: (1) inclusion criteria were English language, human subjects, clinical trial, index medicus, and (2) exclusion criteria were trials involving medications, children, pregnancy, cellular level responses. Twenty studies met the inclusion criteria, reported specific diagnostic threshold levels for AHI, and did not meet any of the exclusion criteria.

Although there were numerous studies on obstructive sleep apnea and CPAP, no randomized, controlled trial specifically evaluated or compared diagnostic criteria in relationship to health outcomes. However, since there were many related clinical trials on OSA and CPAP, indirect evidence on definitions of OSA may be extracted from the methodology of these published trials. Specific diagnostic criteria and treatment thresholds used in the various studies are listed in Table 1.

Table 1. Trials on Continuous Positive Airway Pressure in Sleep Apnea

Author,Year	Design	N c/e*	Diagnosis of OSA for inclusion in Trial	Definition of Moderate to Severe OSA
Ballester et al. 1999	Randomized, controlled trial	104/115	AHI > 15 and severe symptoms (somnolence, daytime impairment,...)	AHI > 15 and severe symptoms, or AHI > 30 + moderate symptoms
Barbe et al. 2001	Randomized, controlled trial	54/55	AHI _ 30	none
d'Ortho et al. 2000	Crossover, cohort	25/25	AHI > 10 events per hour	none

Author,Year	Design	N c/e*	Diagnosis of OSA for inclusion in Trial	Definition of Moderate to Severe OSA
Dimsale et al. 2000	Randomized, placebo controlled trial	39/39	RDI >15	none
Engleman et al. 1999	Randomized, controlled crossover	34/37	Mild sleep apnea, AHI 5 to 15	AHI > 15
Faccenda et al. 2001	Randomized, controlled crossover	68/78	AHI _ 15 and two symptoms	none
Heinzer et al. 2001	Controlled trial	17/20	AHI _ 30	Severe OSA AHI _ 30
Henke et al. 2001	Randomized, controlled trial	32/45	AHI > 10 and daytime sleepiness or AHI > 20	none
Jokic et al. 1998	Controlled, crossover	10/10	AHI > 40	none
Jokic et al. 1999	Randomized, crossover	13/13	AHI > 15	none
Lavie et al. 2000	Population study	2677	Categorization of population into 4 groups	Moderate OSA 30<AHI<51. Severe AHI > 50
Loredo et al. 1999	Randomized, controlled trial	41/48	RDI _ 20	none

Author,Year	Design	N c/e*	Diagnosis of OSA for inclusion in Trial	Definition of Moderate to Severe OSA
Massie et al. 1999	Randomized, crossover	38/47	RDI _ 10	none
Munoz et al. 2000	Controlled trial	135/160	AHI > 20	Mild OSA AHI = 21 Severe OSA AHI = 123
Nieto et al. 2000	Cross-sectional analyses of Sleep Heart Health Study	6132/6440	Categorization of population into 5 groups	Most severe AHI _ 30. Second most severe AHI 15-29.9
Peppard et al. 2000	Prospective cohort	690	AHI _ 15	Moderate to severe AHI _ 15.
Peppard et al. 2000	Prospective cohort	709	Categorization of population	Mild sleep-disordered breathing AHI 5 to 14.9. Moderate to severe AHI _ 15.
Randerath et al. 2001	Randomized, crossover	47/52	AHI _ 10	none
Redline et al. 1998	Randomized, controlled trial	97/111	Mild sleep-disordered breathing, RDI 5-30	none
Shahar et al. 2001	Cross-sectional analyses of SHHS	6424	Categorization of population into 4 groups	Highest quartile AHI > 11. Second highest AHI 4.5-11.0.

Author,Year	Design	N c/e*	Diagnosis of OSA for inclusion in Trial	Definition of Moderate to Severe OSA
Yu et al. 1999	Randomized, controlled trial	34/?	RDI _ 20/hr	none

*sample size completed/enrolled

Of these studies, three population-based or prospective cohort studies have evaluated the association between sleep apnea and hypertension and have estimated the risks. In 2000, Nieto and colleagues reported the findings of cross-sectional analyses on 6132 of the 6440 participants from the Sleep Heart Health Study (SHHS), one of the largest cohort studies.¹⁴ The investigators found that the AHI was associated with the risk of hypertension. The body mass index and demographics adjusted odds ratio (OR) for an AHI of 15-29.9 was 1.32 (95% confidence intervals=1.08-1.61) and 1.60 (95% CI=1.23-2.08). The authors also detected a significant dose-response relationship: as AHI increased, the OR also increased. They concluded that sleep-disordered breathing was associated with systemic hypertension in middle-aged and older individuals. In this study, participants were recruited from ongoing cohort studies of cardiovascular or respiratory disease. Persons with a history of snoring were oversampled to optimize statistical power. Potential biases may have been introduced with the sampling methodology. Generalizability to asymptomatic populations may be reduced. In addition, home polysomnography was used to measure AHI and other parameters.

In 2000, Lavie and colleagues reported the findings of a population study of 2677 adults (aged 20-85) referred for NPSGs with suspected sleep apnea.¹⁵ Using logistic regression analysis, the authors found that the odds of hypertension increased with increasing AHI. The rate of increase in the odds ratio accelerated after an AHI of 20. They concluded that sleep apnea syndrome is associated with hypertension. In this study, all participants had suspected sleep apnea so generalizability may be limited. Also in 2000, Peppard and colleagues reported the results of the Wisconsin Sleep Cohort Study, a prospective study with 709 participants at the four year follow-up period.¹⁶ The authors found that the adjusted odds ratio for an AHI _ 15 was 2.89 (95% CI=1.46-5.64). They concluded that sleep-disordered breathing is likely to be a risk factor for hypertension and consequent cardiovascular morbidity. In this study, the participants were randomly selected from a pool of employees of 4 Wisconsin State agencies who responded to a mailed questionnaire. There was a high drop out rate (40% of original: 480/1189).

In addition, two randomized, controlled trials evaluated CPAP in mild OSA. In 1999, Englemen and colleagues reported the results of a trial to evaluate treatment benefits in terms of daytime symptoms and functioning.¹⁷ Thirty-seven patients with mild sleep apnea/hypopnea syndrome (AHI 5 to 15) were randomly assigned to CPAP or oral placebo for 4 weeks and then crossed over. Of the 37 enrolled, 34 patients completed the study. The authors found that patients who received CPAP had improvements in symptom score ($p<0.01$), subjective sleepiness ($p<0.01$), and performance ($p<0.02$) based on questionnaires and performance testing. In this study, an oral placebo was used so participants were aware of the assignment group. Multiple outcomes were evaluated. Multivariate analysis was not used to adjust for potential confounders.

In 1998, Redline and colleagues reported the results of a randomized, controlled trial to assess the effects of CPAP on mood and functional status. One hundred and eleven individuals with mild sleep-disordered breathing (RDI between 5 and 30) were randomly assigned to either CPAP or conservative therapy (nasal dilator). Of the 111, 97 completed at least 8 weeks of intervention. The authors found that more individuals in the CPAP group had improvements in outcomes compared to conservative therapy ($p<0.05$). They concluded that "CPAP therapy may be beneficial to a broader group of subjects than previously appreciated."¹⁸ In this study, a nasal dilator was used as a control so participants were aware of the assignment group.

Should hypopnea be included in the diagnostic criteria of OSA?

Since the 1980's, hypopnea has been commonly used as a diagnostic criteria for OSA. Gould and colleagues reported that "hypopneas are clinically important" in the consideration of OSA.¹⁹ Whyte and colleagues reported that hypopneas can be scored reproducibly and that there was close agreement (high correlation coefficient) on the number of apneas and hypopneas by two independent polysomnographers.²⁰ However, variations in the definition of hypopnea have been common. In 1994, Moser and colleagues surveyed 45 accredited sleep laboratories and found that "no two laboratories used the same definition and measures of hypopnea."²¹

To assess the current evidence on hypopnea in greater detail, Medline (mid 1998 to 9/2001) was searched using keywords hypopnea and obstructive sleep apnea (OSA). Over 200 studies were found. The literature search was then focused based on the following criteria: (1) inclusion criteria were English language, human subjects, clinical trial, index medicus and (2) exclusion criteria were trials involving medications, children, pregnancy, cellular level responses. Fifteen studies met the inclusion criteria, reported specific definitions, and did not meet any of the exclusion criteria.

Although there were no specific randomized, controlled trials that evaluated the validity of using hypopnea as a diagnostic criterion for OSA in terms of health outcomes, the use of hypopnea appears to have been accepted as current standard of practice. This is apparent in the widespread use of the AHI as documented in the methodology of the relevant trials. However, as noted earlier, variations in the definition of hypopnea are still found. Various definitions are presented in Table 2.

Table 2. Definition of Apnea and Hypopnea

Author,Year	Design	N c/e*	Definition of Apnea and Hypopnea during Sleep
Ballester et al. 1999	Randomized, controlled trial	104/115	Apnea = airflow cessation at least 10s. Hypopnea = airflow reduction at least 10s with 3% decrease oxygen saturation (pulse oximeter).

Author,Year	Design	N c/e*	Definition of Apnea and Hypopnea during Sleep
Barbe et al. 2001	Randomized, controlled trial	54/55	Apnea = respiratory flow cessation > 10s. Hypopnea = significant reduction in respiratory flow followed by arousal or arterial oxygen desaturation greater than 4%.
d'Ortho et al. 2000	Crossover, cohort	25/25	Apnea = absence of breathing > 10s. Hypopnea = airflow < 50% for > 10s.
Engleman et al. 1999	Randomized, controlled crossover	34/37	Apnea = airflow absent for 10s or more. Hypopnea = abdominal or thoracic respiratory movement amplitude of 50% or less or baseline for 10s or more.
Faccenda et al. 2001	Randomized, controlled crossover	68/78	Apnea not defined. Hypopnea defined as >50% reduction in thoracoabdominal mvt.
Heinzer et al. 2001	Controlled trial	17/20	Apnea = cessation of respiratory airflow of at least 10s. Hypopnea = reduction of airflow >50% lasting > 10s.

Author,Year	Design	N c/e*	Definition of Apnea and Hypopnea during Sleep
Lavie et al. 2000	Population study	2677	<p>Apnea = cessation in airflow of at least 10s.</p> <p>Hypopnea = decrease in amplitude of the respiratory signal of at least 50% for a minimum of 10s followed by either a decrease in oxygen saturation of 4% or signs of physical arousal.</p>
Loredo et al. 1999	Randomized, controlled trial	41/48	<p>Apnea = decrements in airflow > 90% for > 10s.</p> <p>Hypopnea = decrements in airflow > 50% but < 90% for >10s.</p>
Massie et al. 1999	Randomized, crossover	38/47	<p>Apnea = cessation of airflow (airflow tracing 0-20% of baseline) > 10s.</p> <p>Hypopnea = reduction of airflow (airflow tracing 20-75%) > 10s.</p>
Meoli et al. for AASM. 2001.	Position paper	-	<p>Apnea = cessation of airflow for 10s or more</p> <p>Hypopnea = abnormal respiratory event at least 10s with at least 30% reduction in thoracoabdominal movement or airflow with at least 4% oxygen desaturation.</p>
		6132	

Author,Year	Design	N c/e*	Definition of Apnea and Hypopnea during Sleep
Nieto et al. 2000.	Cross-sectional analyses of SHHS cohort		<p>Apnea = complete or almost complete cessation of airflow.</p> <p>Hypopnea = decrease in airflow or thoracoabdominal excursion at least 30% for 10s or more + 4% or more desaturation.</p>
Peppard et al. 2000	Prospective cohort	690	<p>Apnea = cessation of airflow lasting 10s or more.</p> <p>Hypopnea = discernible reduction in the sum of thoracic cage plus abdomen respiratory inductance plethysmography amplitude associated with 4% or greater reduction in oxyhemoglobin saturation.</p>
Peppard et al. 2000	Prospective cohort	709	<p>Apnea = cessation of airflow for at least 10s.</p> <p>Hypopnea = discernible reduction in the sum amplitude of the rib-cage plus abdominal excursions on respiratory inductance plethysmography that lasted at least 10s with 4% or greater reduction in oxyhemoglobin saturation.</p>
Shahar et al. 2001	Cross-sectional analyses of SHHS	6424	Respiratory event = decrease in airflow or chest wall movement to an amplitude that was smaller than approx 25% (apnea) or 70% (hypopnea) of the baseline.
Yu et al. 1999	Randomized, controlled trial	34/?	Apnea = decrements in airflow > 90% for >10s.

Author,Year	Design	N c/e*	Definition of Apnea and Hypopnea during Sleep
			Hypopnea = decrements in airflow > 50% but < 90% for >10s.

*sample size completed/enrolled

In addition, the DMERCs have proposed the following definitions: (1) apnea is the cessation of airflow > 10 seconds documented on polysomnogram, (2) hypopnea is a reduction in airflow > 10 seconds associated with a fall in oxygen saturation and an arousal from sleep documented on polysomnogram.²²

Is there scientific evidence of the effectiveness of bilevel positive airway pressure (BiPAP) for the treatment of OSA?

To assess the effectiveness of BiPAP for the treatment of OSA, Medline (1995-9/2001) was searched using keywords bilevel positive airway pressure, BiPAP and obstructive sleep apnea (OSA). Eighteen citations were found. The literature search was focused using the following criteria were used: (1) inclusion criteria were English language, human subjects, clinical trial, index medicus, and (2) exclusion criteria were trials involving medications, children, pregnancy, cellular level responses. One randomized trial met the inclusion criteria and did not meet the exclusion criteria.

In 1995, Reeves-Hoche and colleagues randomized 83 patients to either CPAP (n=52) or BiPAP (n=31) to determine whether BiPAP achieves better patient comfort and hours of use than CPAP. Of the 83 patients enrolled, 62 completed the 1 year study period. The authors found that patient complaints and effective use were similar in both groups but that the dropout rate was significantly higher in the CPAP group (p=0.003). The authors did note a methodologic error in the randomization process resulting in a skewed distribution.²³ This study did not specifically address effectiveness of BiPAP in terms of health outcomes.

Consensus Statements

In 1990, the National Institutes of Health (NIH) convened a consensus development conference and reported that "obstructive sleep apnea is a potentially reversible cause of daytime sleepiness, which may be associated with comorbid conditions and even excess mortality." The panel also noted that "treatment is recommended for more severe degrees of this disorder. Objective indices of severity elicited by polysomnography should include a high index of respiratory disturbances per hour, repetitive episodes of hypoxemia and an abnormally shortened sleep latency. Strict guidelines for therapy have not been adequately validated to dictate thresholds for distinguishing less severely affected patients. At the present time, considerable reliance is made on clinical judgement to initiate a therapeutic trial or regimen."²⁴

In 1994, the American Thoracic Society (ATS) published an official statement on the use of CPAP in sleep apnea syndromes. They reported that "CPAP is effective in the treatment of patients with clinically important obstructive sleep apnea/hypopnea syndrome" and that "CPAP is a safe, effective form of therapy with rare complications." ATS did not present specific diagnostic criteria but noted that "typically patients with greater than 20 apneas or hypopneas/h have been selected for studies examining clinical responses to treatment."

In 1999, Loube and colleagues published a consensus statement and stated that "CPAP treatment is indicated for all OSA patients with an RDI > 30 events per hour, regardless of symptoms, based on the increased risk of hypertension evident from the Wisconsin sleep cohort data." They further stated that "treatment with CPAP is indicated for patients with an RDI of 5 to 30 events per hour accompanied by symptoms of excessive daytime sleepiness, impaired cognition, mood disorders, insomnia, or documented cardiovascular diseases to include hypertension, ischemic heart disease or stroke." They defined apnea as the cessation of airflow > 10s and hypopnea as a recognizable, transient reduction of breathing with > 50% decrease in the amplitude of a validated measure of breathing or a < 50% amplitude reduction that is associated with either an oxygen desaturation of > 3% or an arousal. The authors also reported that "treatment with CPAP is not indicated for asymptomatic patients without cardiovascular diseases who demonstrate mild OSA on diagnostic NPSG (nocturnal polysomnography)." Concerning BiPAP, the authors noted that "a trial of bilevel PAP may be indicated for OSA patients who cannot tolerate CPAP due to persistent massive nasal mask air leakage or discomfort exhaling against positive pressure."²⁵

In 2001, the American Academy of Sleep Medicine (AASM) released a position statement that addressed hypopnea and sleep-disordered breathing. AASM reported that "it is currently standard in clinical practice and epidemiologic studies to assess the severity of sleep-disordered breathing by combining the number of apneas and hypopneas per hour of sleep in an index called the apnea-hypopnea index (AHI) or the respiratory disturbance index (RDI)."²⁶

CMS Analysis

Although there were no specific randomized, controlled trials that evaluated diagnostic criteria of OSA and the use of hypopnea, there were a myriad of clinical trials on CPAP and OSA in general. From this indirect evidence, certain accepted definitions and practices may be identified.

Using the most current clinical trials, it is possible to adopt a set of diagnostic criteria and definitions that are more up to date than the ones used in the 1986 Medicare coverage decision. As in other instances in clinical medicine, the diagnostic criteria of OSA and AHI value for treatment appear to be based more on consensus and standards of practice than direct evidence from clinical trials. This is illustrated in the gradual change of inclusion criteria in clinical trials over the past few years and the convergence of definitions.

The use of hypopnea as a diagnostic criterion has been accepted as a standard of practice.²⁷ This is reflected in the studies shown in Tables 1 and 2, where all protocols included hypopneas. While the use of hypopnea as a diagnostic criterion has been widely accepted, a standard definition of hypopnea has not been crafted. In the 15 clinical trials listed in Table 2, there were several different definitions reported. However, the American Academy of Sleep Medicine has recently proposed a standard definition for hypopnea based on one of the largest trials on OSA, the Sleep Heart Health Study (SHHS).²⁸ In the SHHS, hypopnea was defined as an abnormal respiratory event lasting at least 10 seconds with at least a 30% reduction in thoracoabdominal movement or airflow as compared to baseline, and with at least a 4% oxygen desaturation. This definition incorporates thoracoabdominal movement and desaturation since these two factors have been found to be reproducible and measurable during sleep studies.²⁹ In a letter to CMS, the American Thoracic Society also recommended the same definition of hypopnea.³⁰ Based on the apparent general consensus, CMS will include hypopnea to calculate an apnea-hypopnea index (AHI) for Medicare coverage of CPAP.

In setting a treatment and coverage threshold based on the AHI, two general situations may be considered: patients who do not have symptoms and patients who have symptoms. For patients who do not have symptoms of OSA, treatment with CPAP may still be a consideration given the increased risk for hypertension and related cardiovascular conditions. Three large population-based studies have demonstrated the association between OSA and hypertension. A dose-response relationship between AHI and the risk of hypertension was also reported.^{31,32,33} However, these studies had limitations in sampling, data collection and generalizability. For example, unattended home polysomnography was used in the SHHS but Portier and colleagues found that results from home-based testing may be hampered by missing data or poor quality data.³⁴ Also in studies with very large sample sizes, even small differences in measured outcomes may be found to be statistically significant. In some instances, statistical significance does not necessarily imply clinical significance. Overall, these factors may influence the interpretation of results, especially in borderline or very mild cases of OSA.

In our review of the recent literature, studies have suggested that the risk of hypertension with an AHI > 15 is plausible and clinically important (increased risk of hypertension raised by about 2 fold with wide confidence intervals). The studies are less supportive of a risk with an AHI < 15. Of the 20 trials listed in Table 1, 15 used an AHI > 15 per hour or higher levels. Most used an AHI threshold regardless of symptomatology. Of the 8 studies that reported a severity classification, an AHI > 15 per hour was the lowest value for moderate to severe OSA. Overall, CMS concludes that the use of CPAP to treat OSA for patients with an AHI > 15 without symptoms is based on reasonably strong epidemiologic evidence that this condition increases the risk of hypertension and cardiovascular morbidity.

For patients with symptoms, three randomized, controlled trials have demonstrated that CPAP improves daytime symptoms and functioning, even for patients with mild OSA (AHI > 5).^{35,36,37} Although there were no studies that evaluated discrete health outcomes such as mortality, daytime symptoms such as sleepiness and functioning are important since quality of life and personal safety may be significantly affected. In their consensus statement, Loube and colleagues also supported CPAP treatment for patients with symptomatic, mild OSA (RDI 5 to 30).

Based on these published studies and supported by several evidence-based professional consensus statements, CMS will revise the current Medicare coverage policy to include updated definitions and diagnostic criteria as described below. Since most studies did not identify a severity scale for OSA but instead set an AHI value for treatment with CPAP, CMS will remove the reference to severity levels of OSA for Medicare coverage.

Since both types of devices produce positive airway pressure, BiPAP devices have been considered similar to CPAP devices and appear to have similar indications for use.³⁸ Reeves-Hoche and colleagues found that BiPAP was similar to CPAP in terms of patient complaints and usage but had less treatment discontinuation (dropouts).³⁹ This seems to be reflected in current practices as the use of BiPAP has been reserved for patients who have not responded or tolerated standard CPAP.^{40,41} Since there is insufficient direct evidence on effectiveness of BiPAP in OSA for a national coverage decision, the coverage of BiPAP devices for the treatment of OSA will continue to be determined by the local contractors.

Conclusion

CMS will revise the NCD for CPAP for the treatment of OSA (CIM 60-17) to the following: CPAP will be covered under Medicare in adult patients with OSA if either of the following criteria is met:

(1) AHI > 15, or

(2) AHI > 5 and > 14 with documented symptoms of excessive daytime sleepiness, impaired cognition, mood disorders or insomnia, or documented hypertension, ischemic heart disease or history of stroke.

The AHI is equal to the average number of episodes of apnea and hypopnea per hour and must be based on a minimum of 2 hours of sleep recorded by polysomnography using actual recorded hours of sleep (i.e. the AHI may not be extrapolated or projected). Two hours of recorded sleep is consistent with current practice. Apnea is defined as a cessation of airflow for at least 10 seconds. Hypopnea is defined as an abnormal respiratory event lasting at least 10 seconds with at least a 30% reduction in thoracoabdominal movement or airflow as compared to baseline, and with at least a 4% oxygen desaturation. The polysomnography must be performed in a facility-based sleep study laboratory, and not in the home or in a mobile facility.

1 Guilleminault, 1985.

2 Young, 1993

3 Peppard, 1997.

4 Lavie, 2000.

5 Nieto, 2000.

6 Bradley, 1985.

7 Strollo & Rogers, 1996.

8 Lombard, 1985.

9 Loube, 1999.

10 Gould, 1988.

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